Supplementary methods:

Detection of *H. pylori* infection in gastric samples

Genomic DNA from gastric endoscopic biopsies was extracted by commercially available DNA isolation kit for cells and tissues (Roche Diagnostics GmbH, Germany) as per manufacturer's protocol. The initial *H. pylori* diagnosis was performed by conventional *ureC* and *cagA* gene PCRs using a pair of consensus primers derived from the highly conserved *ureC* (1) and *cagA* intragenic regions (2) while β-globin amplification was used as an internal control as described earlier (3). PCR assays based on *ureC* and *cagA* pre-standardized primer could detect at least 3.6 fg of bacterial DNA which correspond to approximately two *H. pylori* genomes. PCR was performed in a 25µL reaction mixture containing ~100ng DNA, 10mM Tris-HCl (pH 8.4), 50mM KCl, 1.5mM $MgCl_2$, 125 µM of each dNTPs [dATP, dGTP, dCTP, and dTTP (Applied Biosystems, CA, USA)], 5pmol of oligonucleotide primers and 0.5U Taq DNA polymerase [Merck, (Bangalore Genei), India]. Details of PCR primers with respective target region, primer sequence, amplicon size and amplification program are provided in **Supplementary S1 Table**. DNA from known *ureC* and *cagA* gene positive *H. pylori* strain and water blank were assayed invariably in each PCR run as positive and negative controls, respectively. After amplification, 10µl aliquot of PCR products were electrophoresed on 2% agarose gel along with 100 bp DNA ladder as molecular weight marker for analysis of expected amplicon in each sample and visualized under a UV transilluminator and documented in AlphaDigidoc (Alpha Innotech Corp. CA, USA).

Protein isolation from tissue biopsies

Total cellular proteins from biopsies and cell lines were isolated by the method described previously (3, 4). The method involved fine mincing of either fresh or frozen biopsies stored at -80°C, in cold 1×PBS with surgical blade in petridish on ice. The minced tissue material was later centrifuged at 4,000 rpm at 4°C to wash off 1×PBS solution. For preparation of total proteins, the pellet from minced tissue was re-suspended in lysis buffer [20mM Tris (pH 7.4), 250mM NaCl, 2mM EDTA (pH-8.0), 0.1% Triton X-100, 10% protease inhibitor cocktail, 0.4mM PMSF, and 4mM Na_3VO_4] and incubated for 30 min on ice. Lysates were spun at 14,000 rpm for 10 min to remove insoluble material. The concentration of proteins was determined by Bradford method (Bio Rad, California, USA) and the proteins were either used immediately or stored in aliquots at -80°C till further use.

Immunoblotting

To assess cellular levels of survivin and STAT3 in gastric tissues and cell lines, total cellular proteins (50µg/lane) were separated in 8% for STAT3 or 15% for survivin polyacrylamide gel and electrotransferred on PVDF membranes (Millipore Corp, Bedford, MA, USA). The membrane was blocked in PBS containing 5% non- fat skimmed milk and probed with specific antibodies by incubating the membrane overnight in pre-standardized dilution of primary antibodies in blocking solution at 4°C. These blots were washed, incubated with HRP-anti-mouse IgG secondary antibodies and visualized by ECL detection kit (Santa Cruz) followed by exposing the chemiluminescent blot to KODAK X-Omat films (Kodak, India). The membranes were stripped and re-probed for β-actin expression as an internal loading control. The quantitative densitometric analysis of the bands was performed using Alpha Ease FC version

4.1.0 (Alpha Innotech Corporation, IL, USA). The normalized mean fold difference in the integrated densitometric values of bands in the immunoblots was calculated with reference to control that consisted of total cellular proteins isolated from 70% confluent HeLa cells, aliquoted in bulk, stored at -80ºC in preservative.

References:

1. Labigne A, Cussac V, Courcoux P. Shuttle cloning and nucleotide sequences of Helicobacter pylori genes responsible for urease activity. J Bacteriol. 1991;173(6):1920-31.
2. Tummuru MKR, Cover TL, Blaser MJ. Cloning and expression of a high-molecular-mass major antigen of Helicobacter pylori: Evidence of linkage to cytotoxin production. Infection and Immunity. 1993;61(5):1799-809.
3. Shukla S, Shishodia G, Mahata S, Hedau S, Pandey A, Bhambhani S, et al. Aberrant expression and constitutive activation of STAT3 in cervical carcinogenesis: implications in high-risk human papillomavirus infection. Mol Cancer. 2010;9:282.
4. Mahata S, Maru S, Shukla S, Pandey A, Mugesh G, Das BC, et al. Anticancer property of Bryophyllum pinnata (Lam.) Oken. leaf on human cervical cancer cells. BMC Complement Altern Med. 2012;12:15.